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LETTER TO THE EDITOR

Lactobacillus FLORA IN SHORT BOWEL SYNDROME

To The Editor:

In stool of an asymptomatic short bowel syndrome (SBS) patient, Caldarini et al (1) observed an abnormal flora with both marked predominance of two gram-positive anaerobic rods, *Lactobacillus plantarum* and *Lactobacillus salivarius*, and absence of the very common bacteroids. From a continuous culture study they learned that environmental pH affected not only the ecological composition (because of differences in growth rates of the various bacteria), but also the overall production of volatile fatty acids and D- and L-lactic acids. Their results should suggest that increasing the colonic intraluminal pH could be successful in preventing D-lactic acid production.

Over more than four years we have studied longitudinally 14 SBS patients: four infants, three children, and seven adults (2–4). In young SBS children we observed high serum D-lactate and high D-lactate excretion, but in SBS adults the values were rather low (2). We reported about a (diet-associated) circadian rhythm concerning D-lactic acidemia and aciduria in an SBS child (2), and, with this in mind, we discussed the consequences of overnight drip-feeding with regard to D-lactic acidosis (3). The circadian rhythm has been confirmed in an SBS adult; it was less pronounced probably due to presence of D-2-OH-acid dehydrogenase in the adult human body (cf. 5).

In feces of our SBS patients (during both asymptomatic and symptomatic periods), lactobacilli were always the major component of total fecal SBS flora (mean: 70 ± 15%, but sometimes even about 100%; mean in controls: <1%). The resident flora consisted of lactobacilli (10^{10}–10^{12} CFU/g wet feces) and *Escherichia coli* (10^{8}–10^{10} CFU/g wet feces). As transient fecal flora (sometimes up to 10^{12} CFU/g wet feces) were observed only bacteria which are able to produce lactic acid from carbohydrates. In our patients we observed mainly *Lactobacillus acidophilus* and *Lactobacillus fermentum*; in acidotic patients abundancies of these two species (6–8), and of *Lactobacillus casei* (9) and *Lactobacillus buchneri* (6) have been reported. The data of Caldarini et al (1) fit fully with this type of fecal flora with lactobacilli as the major component.

By use of a continuous culture study, Caldarini et al (1) confirmed that pH plays an important role in bacterial ecology and thus also in intestinal ecology. The acidophilic lactobacilli are strongly favored by rather low pH (4–5.5), and the nonacidophilic enterobacteria by rather high pH (6–7). All lactobacilli produce D- and/or L-lactic acid; however, many enterobacteria produce lactic acid, and a variety of short chain fatty acids (SCFA). Therefore, it is not surprising that environmental pH may indirectly affect production of these acids (1). Under anaerobic conditions some lactobacilli [such as *L. plantarum* (10)] are excellent producers of both D- and L-lactic acid from fermentable sugars, whereas other lactobacilli produce only D-lactic acid, or [like *L. salivarius* (10)] L-lactic acid. Maximal L-lactate production at pH 5.5 probably reflects *L. salivarius* being less acidophilic than *L. plantarum*. However, the experimental data of Caldarini et al (1) confirm that a rather low intestinal pH (4–5.5) in SBS patients favors high lactate production by lactobacilli.

Although Caldarini et al did not observe streptococci in the SBS feces (1), probably due to low fecal pH and/or antimicrobial factors produced by fecal flora, numerous streptococci are ingested daily by humans. Anaerobic growth of these bacteria (mainly L-lactate producers) is favored especially by intermediate pH (5–6). Thus, after increasing the colonic intraluminal pH, the final SBS flora may differ strongly from the flora obtained after raising the pH in a continuous culture as done by Caldarini et al (1). Apart from how to create a constant rather high colonic intraluminal pH, such an increase will lower the pH barrier for other less acidophilic bacteria (enterobacteria, staphylococci, and pseudomonads), which are nearly all more pathogenic than lactobacilli.

L-Lactate is an excellent substrate in our tissues, and lactobacilli are intrinsic inhabitants of SBS intestines. Therefore, we propose [apart from dietary measures (8)] total replacement of the D- and L-lactate producing intestinal flora (at least in SBS children) by probiotic, L-lactate-only producing lactobacilli, such as *Lactobacillus casei* subsp. *casei* or *Lactobacillus casei* subsp. *rhamnosus* (10), to reduce D-lactic acidemia and acidosis in SBS. In SBS adults probiotic D-/L-lactate-producing *L. acidophilus* may suffice due to presence of human D-2-OH-acid dehydrogenase (5).

G.P.A. BONGAERTS, PHD
Department of Medical Microbiology
J.J.M. TOLBOOM, MD PHD
Department of Pediatrics
A.H.J. NABER, MD
Department of Gastroenterology and Hepatology
J.A.J.M. BAKKEREN, PHD
Laboratory of Pediatrics
R.S.V.M. SEVERIJNEN, MD
Department of Pediatric Surgery
J.L. WILLEMS, PHD
Department of Clinical Chemistry
University Hospital of Nijmegen St. Radboud
Nijmegen, The Netherlands
W.J.K. SPERL, MD PHD
Kinderspital
Landeskrankenanstalten Salzburg
Salzburg, Austria

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